Increased ethnicity and socioeconomic data collection required in stroke associated with COVID-19

For over 4 months, we have been in the privileged position of witnessing real-time trends in the literature via our weekly curation of The Neurology and Neuropsychiatry of COVID-19 blog. There is an emerging recognition of the need to report the severity and outcomes of neurological complications of SARS-CoV-2 infection. Stroke is a rare but well-documented complication, with most prevalence estimates varying slightly around 0.8%. It is therefore important to elucidate both those at risk of developing stroke and stratifying those at risk of more severe outcomes when stroke occurs.

We were greatly interested to read the letter by Dmytriw et al, which highlighted potential racial disparities in outcomes from ischaemic stroke after COVID-19. Their novel study built on outcomes from ischaemic stroke after COVID-19 had diabetes (63% vs 28.6%) and higher levels of low-density lipoproteins (37.7% greater) in general. Diabetes is more common in people from black backgrounds than in the general population, and it is well recognised that comorbidities confer higher risk of stroke and mortality. Accordingly, one possibility is that an increase in stroke mortality rates in BAME groups may simply reflect an increased prevalence of comorbidities such as diabetes or higher cardiovascular risk.

A second possibility is that race and ethnicity may interact with comorbidity and lower socioeconomic background to limit access to care. Both Dmytriw et al and Katz et al proposed social determinants of health, access to care and geographical differences as potential mediators of the increased incidence and mortality of COVID-19-related stroke. Stakeholders interviewed by Public Health England argue further that COVID-19 has exacerbated existing health inequalities and highlight the lack of targeted programmes for chronic disease prevention in these groups. Although explanatory mechanisms remain unclear, we noted in passing the lower prevalence of thrombectomy and use of tissue plasminogen activator in African-Americans in Dmytriw et al’s cohort.

Great caution is required in interpreting racial discrepancies in outcomes from COVID-19. The influence of ethnicity is likely to be complex and multifactorial. Renieri drew on broader COVID-19 research on increased mortality associated with black race to suggest that after adjusting for various socioeconomic factors, black race with COVID-19 was not associated with increased in-hospital mortality. Although this finding goes some way to exploring potential underlying mechanisms driving racial disparities, it may yet miss mediators of mortality associated with stroke in COVID-19 specifically or influences on higher viral transmission among black individuals.

In future, for instance, studies might hypothesise an interaction between these outcomes and overcrowded housing, greater reliance on public transport, living in dense population centres, occupational risks and/or socioeconomic inequalities.

Katz et al proposed further that differences in risk factor modification, diet and genetic predisposition may also contribute to health outcome inequality. We commend the authors of these studies for highlighting these issues and the potential reasons behind any racial disparity. It is notable, however, that both Katz et al’s and Dmytriw et al’s studies collected data from North America. Differing ethnic profiles, healthcare systems and the impacts of racial discrimination between North America and other countries make it difficult to generalise any social and structural effects on racial disparities in COVID-19 infection and stroke but are useful in highlighting a gap in the global literature.

Therefore, we wish to propose that alongside collecting and recording comprehensive ethnicity data, prospective stroke registers and studies in COVID-19 broaden globally beyond simply measuring race. Socioeconomic background, occupation, lifestyle, housing circumstances, access and attitudes to care, and feelings of discrimination may all help shed light on mechanisms for potential racial disparity. This added granularity of detail will help build high-quality evidence to unpick the complex effect of race on health inequalities driving differential racial outcomes in stroke after COVID-19.

Danish Hafeez, Jia Song, Cameron Watson, Alasdair Rooney, Timothy R Nicholson, The SARS-COV-neuro collaboration

1 School of Medicine, The University of Manchester, Manchester, UK
2 Dean Cross Personality Disorder Service, East London NHS Foundation Trust, London, UK
3 Preventive Neurology Unit, Wolfson Institute of Preventive Medicine, London, UK
4 Centre for Clinical Brain Sciences, The University of Edinburgh, Edinburgh, UK
5 Section of Cognitive Neuropsychiatry, Institute of Psychiatry, London, UK

Correspondence to Danish Hafeez, The University of Manchester, Manchester M13 9PL, UK; hafeez78.dh@gmail.com

Twitter Danish Hafeez @danish_hafeez1, Timothy R Nicholson @Tim_R_Nicholson and The SARS-COV-neuro collaboration @Neuropsychcoid

Collaborators The SARS-COV-neuro collaboration: Ben Cross, Benedict Michael, Emma Rengasamy, Hamilton Morrin, Jamie Badenoch, Lucretia Thomas, Mao Fong Lim, Mark Ellul, Matt Butler, Tom Pollak, Susannah Pick.

Contributors DH came up with the initial idea. DH, JS, CW, AR and TRN all contributed to the final manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not required.
To cite Hafeez D, Song J, Watson C, et al. J Neurol Neurosurg Psychiatry Epub ahead of print: [please include Day Month Year]. doi:10.1136/jnnp-2020-325057
Received 8 September 2020
Accepted 9 September 2020
J Neurol Neurosurg Psychiatry 2020;0:1–2.
doi:10.1136/jnnp-2020-325057

ORCID iD
Danish Hafeez http://orcid.org/0000-0003-3712-136X

REFERENCES
4 England PH. Beyond the data: understanding the impact of COVID-19 on BamE groups. PHE publications 2020.